V Arechavala-Gomeza

List of Publications by Year
in descending order

58	3,268	304368	301761
papers	citations	22 h-index	39 g-index

61	61	61	3257
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Exon skipping and dystrophin restoration in patients with Duchenne muscular dystrophy after systemic phosphorodiamidate morpholino oligomer treatment: an open-label, phase 2, dose-escalation study. Lancet, The, 2011, 378, 595-605.	6.3	803
2	Local restoration of dystrophin expression with the morpholino oligomer AVI-4658 in Duchenne muscular dystrophy: a single-blind, placebo-controlled, dose-escalation, proof-of-concept study. Lancet Neurology, The, 2009, 8, 918-928.	4.9	617
3	Delivery of oligonucleotideâ€based therapeutics: challenges and opportunities. EMBO Molecular Medicine, 2021, 13, e13243.	3.3	181
4	Comparative Analysis of Antisense Oligonucleotide Sequences for Targeted Skipping of Exon 51 During Dystrophin Pre-mRNA Splicing in Human Muscle. Human Gene Therapy, 2007, 18, 798-810.	1.4	144
5	Muscle histology vs MRI in Duchenne muscular dystrophy. Neurology, 2011, 76, 346-353.	1.5	134
6	Dystrophin quantification and clinical correlations in Becker muscular dystrophy: implications for clinical trials. Brain, 2011, 134, 3547-3559.	3.7	125
7	Delivery is key: lessons learnt from developing spliceâ€switching antisense therapies. EMBO Molecular Medicine, 2017, 9, 545-557.	3.3	119
8	A Duchenne Muscular Dystrophy Gene Hot Spot Mutation in Dystrophin-Deficient Cavalier King Charles Spaniels Is Amenable to Exon 51 Skipping. PLoS ONE, 2010, 5, e8647.	1.1	102
9	Restoration of the Dystrophin-associated Glycoprotein Complex After Exon Skipping Therapy in Duchenne Muscular Dystrophy. Molecular Therapy, 2012, 20, 462-467.	3.7	99
10	Chronic Systemic Therapy With Low-dose Morpholino Oligomers Ameliorates the Pathology and Normalizes Locomotor Behavior in mdx Mice. Molecular Therapy, 2011, 19, 345-354.	3.7	97
11	Revertant fibres and dystrophin traces in Duchenne muscular dystrophy: Implication for clinical trials. Neuromuscular Disorders, 2010, 20, 295-301.	0.3	80
12	Immunohistological intensity measurements as a tool to assess sarcolemmaâ€associated protein expression. Neuropathology and Applied Neurobiology, 2010, 36, 265-274.	1.8	77
13	Stakeholder cooperation to overcome challenges in orphan medicine development: the example of Duchenne muscular dystrophy. Lancet Neurology, The, 2016, 15, 882-890.	4.9	77
14	Antisense Oligonucleotide-Mediated Exon Skipping for Duchenne Muscular Dystrophy: Progress and Challenges. Current Gene Therapy, 2012, 12, 152-160.	0.9	73
15	Dystrophin quantification. Neurology, 2014, 83, 2062-2069.	1.5	73
16	Biochemical Characterization of Patients With In-Frame or Out-of-FrameDMDDeletions Pertinent to Exon 44 or 45 Skipping. JAMA Neurology, 2014, 71, 32.	4.5	71
17	An Overview of Alternative Splicing Defects Implicated in Myotonic Dystrophy Type I. Genes, 2020, 11, 1109.	1.0	66
18	Comparative analysis of antisense oligonucleotide sequences targeting exon 53 of the human DMD gene: Implications for future clinical trials. Neuromuscular Disorders, 2010, 20, 102-110.	0.3	44

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19	Exon Skipping Quantification by Quantitative Reverse-Transcription Polymerase Chain Reaction in Duchenne Muscular Dystrophy Patients Treated with the Antisense Oligomer Eteplirsen. Human Gene Therapy Methods, 2012, 23, 336-345.	2.1	38
20	Splicing modulation therapy in the treatment of genetic diseases. The Application of Clinical Genetics, 2014, 7, 245.	1.4	33
21	The contribution of human synovial stem cells to skeletal muscle regeneration. Neuromuscular Disorders, 2010, 20, 6-15.	0.3	32
22	Utrophin modulator drugs as potential therapies for Duchenne and Becker muscular dystrophies. Neuropathology and Applied Neurobiology, 2021, 47, 711-723.	1.8	22
23	Why dystrophin quantification is key in the eteplirsen saga. Nature Reviews Neurology, 2018, 14, 454-456.	4.9	20
24	Researcher's Perceptions on Publishing â€œNegativeâ€•Results and Open Access. Nucleic Acid Therapeutics, 2021, 31, 185-189.	2.0	19
25	The Biomarker Potential of miRNAs in Myotonic Dystrophy Type I. Journal of Clinical Medicine, 2020, 9, 3939.	1.0	15
26	A multicenter comparison of quantification methods for antisense oligonucleotide-induced DMD exon 51 skipping in Duchenne muscular dystrophy cell cultures. PLoS ONE, 2018, 13, e0204485.	1.1	14
27	Myoblots: dystrophin quantification by inâ€cell western assay for a streamlined development of Duchenne muscular dystrophy (DMD) treatments. Neuropathology and Applied Neurobiology, 2018, 44, 463-473.	1.8	12
28	Duchenne muscular dystrophy cell culture models created by CRISPR/Cas9 gene editing and their application in drug screeningÂ. Scientific Reports, 2021, 11, 18188.	1.6	10
29	Joining European Scientific Forces to Face Pandemics. Trends in Microbiology, 2021, 29, 92-97.	3.5	5
30	Antisense RNA Therapeutics: A Brief Overview. Methods in Molecular Biology, 2022, 2434, 33-49.	0.4	5
31	Evaluation of Exon Skipping and Dystrophin Restoration in In Vitro Models of Duchenne Muscular Dystrophy. Methods in Molecular Biology, 2022, 2434, 217-233.	0.4	5
32	Chapter 14 Familial amyotrophic lateral sclerosis. Handbook of Clinical Neurology / Edited By P J Vinken and G W Bruyn, 2007, 82, 279-300.	1.0	3
33	Exon-skipping therapy for Duchenne muscular dystrophy â€“ Authors' reply. Lancet, The, 2012, 379, e10-e11.	6.3	2
34	COST Actions: fostering collaborative research for rare diseases. Lancet Neurology, The, 2019, 18, 989-991.	4.9	2
35	Sharing â€œNegativeâ€•Results in Neuromuscular Research: A Positive Experience. Journal of Neuromuscular Diseases, 2021, 8, 765-767.	1.1	2
36	T.P.1 02 A phase I/II clinical trial in Duchenne muscular dystrophy using IM and IV delivered antisense oligonucleotides: The MDEX consortium. Neuromuscular Disorders, 2006, 16, 685.	0.3	1

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37	T.O.3 Restoration of dystrophin expression in Duchenne muscular dystrophy: A single blind, placebo-controlled dose escalation study using morpholino oligomer AVI-4658. Neuromuscular Disorders, 2009, 19, 659.	0.3	1
38	Measuring dystrophinâ€"faster is not necessarily better. Nature Reviews Neurology, 2012, 8, 469-469.	4.9	1
39	G.P.12.11 Do revertants increase with age in Duchenne muscular dystrophy boys?. Neuromuscular Disorders, 2007, 17, 842.	0.3	0
40	G.P.6.01 Establishing the parameters for clinical trials of antisense oligonucleotide therapy in Duchenne muscular dystrophy. Neuromuscular Disorders, 2008, 18, 773.	0.3	0
41	T.P.4.09 Measuring restored dystrophin in treated muscle: An immunohistological intensity measurement method. Neuromuscular Disorders, 2009, 19, 615.	0.3	0
42	P3.08 Induction of dystrophin in DMD patients by antisense oligonucleotide AVI-4658 restores the dystrophin glycoprotein complex. Neuromuscular Disorders, 2010, 20, 643.	0.3	0
43	O04 Results of a systemic antisense study in Duchenne muscular dystrophy. Neuromuscular Disorders, 2010, 20, S2.	0.3	0
44	P03 The characterisation of out of frame duplications in DMD patients. Neuromuscular Disorders, 2010, 20, S5.	0.3	0
45	P05 Induction of dystrophin in Duchenne muscular dystrophy patients by antisense oligonucleotide AVI-4658 restores the dystrophin-associated glycoprotein complex. Neuromuscular Disorders, 2010, 20, S6.	0.3	0
46	P27 Chronic long term administration of phosphorodiamidate morpholino oligomer profoundly ameliorates activity, muscle strength and phenotype in dystrophic mdx mice. Neuromuscular Disorders, 2010, 20, S12.	0.3	0
47	P03 Exon skipping and dystrophin restoration in Duchenne muscular dystrophy patients after systemic phosphorodiamidate morpholino oligomer treatment. Neuromuscular Disorders, 2011, 21, S7-S8.	0.3	0
48	P01 Correlation of internally deleted dystrophin and dystrophin-associated protein expression with clinical severity in Becker muscular dystrophy. Neuromuscular Disorders, 2012, 22, S7.	0.3	0
49	P10 The next DMD exon skipping trial: selection of AO target. Neuromuscular Disorders, 2012, 22, S9-S10.	0.3	0
50	T.P.31 Biochemical and clinical variability of Becker muscular dystrophy: Predicting optimal target exons for exon skipping therapy in Duchenne muscular dystrophy. Neuromuscular Disorders, 2012, 22, 862.	0.3	0
51	P16 Towards a consensus on biochemical outcome measures for Duchenne muscular dystrophy clinical trials. Neuromuscular Disorders, 2014, 24, S11.	0.3	0
52	Quantifying dystrophin in cell culture: A method to accelerate preclinical assessment of DMD treatments. Neuromuscular Disorders, 2015, 25, S254.	0.3	0
53	Myo-cytoblots: Quantification of dystrophin by in-cell western assay for a streamlined development of DMD treatments. Neuromuscular Disorders, 2016, 26, S159.	0.3	0
54	Selection of reference genes for normalisation of dystrophin mRNA RT-qPCR data. Neuromuscular Disorders, 2016, 26, S159-S160.	0.3	0

#	Article	IF	Citations
55	P.291Overcoming barriers to establish a CRISPR/Cas9 edition protocol for human myoblasts. Neuromuscular Disorders, 2019, 29, S152.	0.3	0
56	P.290Dystrophinopathic subjects with a specific mega-deletion of exons 45-55 in the DMD gene, as a template for CRISPR/Cas9 therapy in Duchenne muscular dystrophy. Neuromuscular Disorders, 2019, 29, S151-S152.	0.3	0
57	Special Issue â€œGenetic Advances in Neuromuscular Disorders: From Gene Identification to Gene Therapyâ€: Genes, 2021, 12, 242.	1.0	0
58	Lessons learned from developing an oligonucleotide drug for a rare disease. , 2022, , 121-137.		0